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UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
REGION 10
OREGON OPERATIONS OFFICE
811 S.W. 6th Avenue
Portland, Oregon 97204

March 24, 2006

Mr. Jim McKenna
Port of Portland & Co-Chairman, Lower Willamette Group
121 NW Everett
Portland, Oregon 97209

Mr. Robert Wyatt
Northwest Natural & Co-Chairman, Lower Willamette Group
220 Northwest Second Avenue
Portland, Oregon 97209

Re: Portland Harbor Superfund Site; Administrative Order on Consent for Remedial Investigation and Feasibility Study; Docket No. CERCLA-10-2001-0240. Ecological Preliminary Risk Evaluation

Dear Messrs. Wyatt and McKenna:

EPA has completed its review of the document titled, "Ecological Preliminary Risk Evaluation (PRE). This document was submitted on behalf of the Lower Willamette Group (LWG) by Windward Environmental LLC on September 9, 2005. EPA comments are attached.

The PRE focuses on a screening level evaluation of risk based on a tissue residue and dietary approach, evaluates the relationship between chemicals of interest (COI) in sediment and tissue to determine whether BSAFs can be derived, identifies initial chemicals of potential concern (COPCs) and identifies data gaps that may be filled through subsequent investigations prior to the baseline ecological risk assessment (ERA).

Although the PRE provides useful information about the chemicals that contribute to risk, the conclusions presented in the PRE are of limited usefulness for the following reasons:

1. The PRE does not evaluate all data collected to date – The PRE only evaluates chemicals detected in Round 1 Tissue and Sediment and in the initial round of surface water sampling (November 2004). Although Round 2 sediment data was utilized in the dietary assessment, chemicals detected in Round 2 but not Round 1 were not included in the analysis. In addition, data collected during 2005 was not included in the analysis (e.g., benthic tissue and co-located sediments which will be useful for developing site specific biota-sediment accumulation factors – BSAFs)
2. The PRE focuses on maximum concentrations and site wide averages – The analysis presented in the PRE is useful for screening chemicals but does not address the issue of spatial scale and identification of those areas that pose the greatest risk to ecological receptors.

3. The PRE does not consider all exposure pathways – The PRE does not consider key exposure pathways such as the risk to benthic community (as measured through bioassays) and the risks associated with groundwater discharge.
4. The PRE does not consider recent refinements to the risk assessment approach – The PRE does not consider revisions to the ecological risk assessment as described in EPA's December 2, 2005 Identification of Round 3 Data Gaps Memorandum, EPA's February 17, 2006 Scope of Work or the LWG's March 17, 2006 Proposed Ecological Risk Assessment Decision Framework
5. The PRE may be superseded by the Round 2 Comprehensive Site Characterization Summary and Data Gaps Analysis Report – The Programmatic RI/FS Work Plan requires that the Round 2 Comprehensive Site Characterization Summary and Data Gaps Analysis Report include site specific preliminary remediation goals as well as a more thorough evaluation of the distribution of contaminants and concomitant risk at the site. This evaluation is expected to be far more thorough than the evaluation presented in the PRE.
6. The PRE may be superseded by the Round 3 Data Collection Efforts – The Round 3 characterization effort is expected to include the collection of additional biota tissue. This tissue data may alter some of the conclusions presented in the PRE.

For the above reasons, EPA does not recommend revision and resubmittal of the PRE. Rather the attached comments should be incorporated as appropriate into the Round 2 Comprehensive Site Characterization Summary and Data Gaps Analysis Report . EPA expects that ongoing discussions regarding the ecological risk assessment for the Portland Harbor Site in conjunction with the initial evaluation of risks as presented in the PRE will provide greater clarity regarding the baseline ecological risk assessment.

Please contact Chip Humphrey at (503) 326-2678 or Eric Blischke (503) 326-4006 if you have any questions. All legal inquiries should be directed to Lori Cora at (206) 553-1115.

Sincerely,

Chip Humphrey
Eric Blischke
Remedial Project Managers

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EPA Comments on Ecological Preliminary Risk Evaluation
March 24, 2006

Introduction:

The stated purpose of the Ecological Preliminary Risk Evaluation (PRE) is "to provide a preliminary and protective analysis based on available information to help identify which chemicals have the potential to drive ecological risk, which chemicals have negligible potential for driving ecological risks and which chemicals and exposure pathways need additional data to adequately assess ecological risk and management questions for the site." The PRE focuses on evaluating risk based on a tissue residue and dietary approach, evaluates the relationship between chemicals of interest (COI) in sediment and tissue to determine whether BSAFs can be derived, identifies initial chemicals of potential concern (COPCs) and identifies data gaps that may be filled through subsequent investigations prior to the baseline ecological risk assessment (ERA).

General Comments:

Overall, the PRE does a good job at presenting site-wide risk to species that feed on fish and are exposed to harbor wide prey items (e.g. piscivorous birds and mammals). However, the assessment of receptors that would not be expected to be exposed to an ISA wide mean concentration, which include some fish (e.g. sculpin, smallmouth bass), and invertebrates is limited in that it relies on site-wide maximums. It ~~, and~~ therefore identifies those contaminants that do not exceed the highest concentration detected. This may be useful for screening purposes, but the use of site-wide maximums (and site-wide 95% upper confidence limits – UCLs) limits the conclusions that may be drawn from this evaluation.

The PRE is most useful for identifying COPCs for higher trophic level organisms (e.g. birds and mammals), and can help screen out contaminants that are not likely contaminants of concern. The exception to this is for bioaccumulative? contaminants that were not measured in round 1 tissue sampling, such as PBDEs. For the evaluation of fish health, this evaluation will be better informed after additional tissue samples are taken with different compositing methodology in order to evaluate more localized effects. The addition of benthic tissue (e.g., clam and Lumbriculus testing), as well as the use of sediment EPCs that are calculated with the home range and likely habitat of the receptor in mind, will help reduce these uncertainties.

The evaluation presented in the PRE is based on Round 1 fish tissue data. Data collected as part of 2005 Round 2 data collection efforts (e.g., benthic tissue) and future Round 3 data collection efforts may alter the results of the screening analysis.

The presentation of data is focuses on site-wide risk and not site-specific risk (localized areas). Minimums and maximums are presented with no location numbers, and no information is provided on how many of the data points fall above a TRV range (e.g. is there one or many samples that are above a given TRV?). Evaluation of data in the Round 2 Comprehensive Site Summary and Data Gaps Analysis Report should build on the analysis presented in the PRE and present maps depicting the location of TRV exceedances and exceedance frequency information.

This initial PRE has focused on maximum sediment / tissue concentrations and 100% site use. Explorations moving away from those assumptions are said to "be explored" in the Round 2 Comprehensive Site Summary and Data Gaps Analysis Report. As presented in the March 15, 2006 Ecological Risk Assessment Decision Framework, exposure occurs over a range of scales based on the life history of the receptor. These scales range from a point by point exposure estimate for receptors with small home ranges to intermediate scale for receptors such as smallmouth bass, to the entire Portland Harbor site for species with large home ranges. Further discussion regarding the scale of exposure is required prior to completion of the baseline ERA.

Page 6, Section 2.3, Identification of Exposure Pathways: The text states that the following complete and major pathways will be evaluated quantitatively in the BERA: Aquatic plants, benthic invertebrates, fish, amphibians and reptiles, and birds and mammals. Transition zone water is not mentioned as a pathway for the organisms mentioned. The pathways should include direct contact / uptake from transition zone water for some receptors as presented in Figure 4 (Ecological Risk Assessment Conceptual Site Model) of EPA's December 2, 2005 Identification of Round 3 Data Gaps Memorandum (Data Gaps Memo). EPA recognizes that the ecological risk assessment approach is presented in many documents submitted at various times and that further discussion about the Ecological Risk Assessment is underway. However, at the end of the day, pathway evaluations must be consistent among documents. For example, the DRAFT food web model submittal has several fish ventilating pore water, and in this document it is stated that those pathways are incomplete (or won't be evaluated).

Page 7, Section 2.4, Receptor Groups and Exposure Pathways: The footnote here indicates that the primary line of evidence for benthic invertebrates would be the bioassay results indicating direct toxicity. However, we have multiple lines of evidence because that evaluation does not evaluate all relevant pathways. These include:

- Chemicals that would be lost during the bioassay and sediment testing, including VOCs.
- Static bioassay tests do not represent exposure occurring from fluxing / discharging groundwater. The primary line of evidence should be the comparison of transition zone water in these areas to aquatic benchmarks such as AWQCs.
- Chemicals with high Kow values would not reach equilibrium during the test durations of the bioassay test, but may accumulate and elicit effects including (e.g. PAHs and organochlorine chemicals). A primary line of evidence for these chemicals should be the tissue residue approach.

Further discussion of the lines of evidence approach is expected in conjunction with EPA's review of the March 145, 2006 Ecological Risk Assessment Decision Framework.

Page 8, Section 2.5, Footnote 4: The footnote states that only sediment data from Round 1 sampling was included to select COIs. The list of additional COIs identified in Round 2 sediment is presented in table E-1 in Appendix E. This point should be mentioned in other parts of the report as well to clarify that the PRE of Round 1 and Round 2 data was limited to those chemicals detected in Round 1.

In addition, it is unclear how were PCB sums were calculated. For example, if certain Aroclors were not detected in Round 1 sampling, but were in Round 2, were they included in the PCB sums for sediment (e.g. in calculating dietary exposure to fish, birds and mammals)? Table 2-2

shows the COIs identified for each receptor group based on this analysis. However, the total PCB values are footnoted indicating that limited Aroclors were detected in Round 1 sampling. Does this mean that Aroclors contributing to total PCB concentrations (from Round 2) in sediment were not used in this analysis (e.g. 1221 and 1268)?

The italicized comments below relate to data presentation. It is unclear whether the PRE will be revised or if this information should more appropriately be presented in the Round 2 Comprehensive Report.

Page 10, Section 3.1.1 and 3.1.2, Surface Sediment and Tissue Data: *For understanding localized risk, the summary of the chemistry and tissue results (Appendix D) should include location numbers (at least where the min and max were detected).*

Page 10, Section 3.1.2, Footnote 5: *Locations of fish collections should be presented, and for all fish species (e.g. carp). Summary maps can easily be created from the characterization reports.*

Page 11, Section 3.1.3, Surface Water Data: PCB concentrations were not available to estimate aquatic invertebrate tissue concentrations for use in the dietary exposure estimates. Since the XAD analysis is a must for estimating tissue, this is a limitation in dietary estimates. They likely underestimate PCB concentrations, as the peristaltic pumps did not achieve the necessary detection limits.

Need to figure out where to put this comment. Page 13, Section 3.2.2, Tissue Data: *In the Baseline Ecological Risk Assessment (BERA), surface water concentrations of contaminants in the AOC should be compared to concentrations identified in the literature as impacting adult or juvenile salmon and lamprey via olfactory disruption, and a risk analysis should be made based on the water concentrations in the harbor.*

Page 14, Section 3.3.2, Last Paragraph, Summation Rules: This document is using the "Guidelines for Data Reporting" (Kennedy/Jenks et. al 2004). However the PRE also utilizes summation rules as described in this section (but not presented in the PRE Approach TM). The PRE states that for each sampling location where "some analytes contributing to the sum are detected and some are not detected, only detected concentrations will be summed to represent the total concentration." Summation rules should allow for the inclusion of non-detects at ½ the detection limit. Summation rules should not be used where one of the individual isomers is more toxic than the others in the sum. This could over or underestimate the risk depending on the composition. For example, alpha endosulfan has been shown to be about three times as toxic as the beta isomer of endosulfan. This could be addressed in a conservative screening manner if the TRV of the most toxic isomer was utilized in the screening step. It should be noted that comparing total concentrations of metabolites or transformation compounds will typically overestimate risk and would not be appropriate when documenting toxicological effects have been reported for a specific compound. This method should not be used in the BERA for certain contaminant-receptor pairings, such as when evaluating the risks of DDE (i.e., not total DDTs) to birds using the egg method.

Avian-based toxicity equivalent factors (TEFs) are generally accepted by the scientific community and should be used when calculating toxic equivalents (TEQs) for a bird receptor. The TEFs for birds should be considered conservative because they are largely derived from evaluation of chickens, which are very sensitive to dioxin-like compounds compared to other organisms. It is unclear which TEFs were used to evaluate birds in the PRE, but it is inappropriate to use mammalian-TEFs to evaluate TEQs for birds. TEQs derived for birds should be based on avian-TEFs in subsequent documents. Also, it appears that non-detected concentrations of dioxin-like compounds where given a value of ½ the reporting limit when calculating TEQs. This calculation is overly conservative; dioxin-like compounds that are below reporting limits should not be used in the TEQ calculation (reporting limits should be noted and flagged if they are elevated over expected values when reported TEQ results).

Other questions regarding the summation rules include:

- Applicability of summation varies with the receptor and pathway as well as the toxicity of the individuals in the sum. The text is not specific, does this applies to water, sediment or tissue, or all media?
- The text states, "total concentrations were calculated for Round 1 and Round 2 and relevant non-LWG data collected data sets per sampling location". What non-LWG datasets were included and which were not? What criteria were used?
- In addition to the SCRA database, do we need to review an "eco risk SCRA" database? Is this available?

Page 14, Section 3.3.3, Reduction Rules for Existing Data: Data reduction rules consistent with the data reduction rules for LWG generated data should be developed. Unless there is a compelling need, different rules should not be applied to different data sets.

Page 15, Section 4.0, Effects Characterization, COIs with no TRVs: *TRVs were not developed aluminum and manganese. They proposed to look at available upstream and /or state region-wide background concentration. This analysis should be presented here, as we have some of the data now. If they are elevated, we need to develop a plan for assessment (e.g. the development of TRVs). Need to address Mn and Al background issue.*

Page 16, Section 4.1.1, Aquatic Tissue Residue TRVs, Tier 1: The PRE describes a two-tiered approach to develop tissue residue TRVs for aquatic life that are appropriately conservative for use in a screening level assessment. In Tier 1, the tissue residue TRV for a chemical of interest (COI) was based on the 5th percentile of the species sensitivity distribution (SSD) if 20 or more toxicity values were available. If not, the 5th percentile LOEC reported in Dyer et al. (2000) was identified as the TRV. A Tier 2 approach was used for those COIs with less than 20 lowest observed effect concentrations (LOECs) available, or if no 5th percentile toxicity value was available from Dyer et al. (2000). In Tier 2 of the PRE, the tissue residue TRV for a COI was calculated as the product of the EPA's ambient water quality criterion (AWQC) multiplied by a bioconcentration factor (BCF). Ultimately, therefore, the PRE followed a hierarchy in which tissue residue TRVs for aquatic organisms were based on (1) the 5th percentile LOEC when more than 20 LOECs were available, (2) the 5th percentile LOEC from Dyer et al. (2000), and (3) the AWQC multiplied by a BCF.

However, the approach used by the LWG in the PRE does not appear to be entirely consistent with EPA's comments on the Provisional TRV Technical Memorandum. In Tier 1, "EPA emphasizes that use of the TRVs in Dyer et al. (2000) is in addition to, not instead of the TRVs for fish, bivalves and crayfish developed by the LWG during their extensive literature review and presented in the Provisional TRV Technical Memorandum." Thus, it appears that there should not be a hierarchy of decisions in Tier 1, but, rather, the Dyer et al. 5th percentile LOECs should supplement the TRVs developed by the LWG from their detailed literature reviews. The other apparent inconsistency between the PRE and EPA's direction relates to the Tier 2 approach. If a TRV could not be developed for a COI in Tier 1, the PRE automatically defaulted to basing the TRV on the AWQC multiplied by a BCF. The EPA's direction, however, was to also consider the number of LOECs identified from the detailed literature search. If 8-19 LOECs were available, either the lower value of the lowest LOEC or AWQC multiplied by a BCF is to be used in developing a Tier 2 TRV. It does not appear that the PRE followed this step.

It appears that the TRV was selected from the 5^{th} percentile of LOEC data only from fish, clams and crayfish and not ALL aquatic species LOEC data. It is important that other invertebrate data is included in the distribution. The text here is a little unclear, but Table 4-1 footnote B indicates only fish, clam and crayfish data were used. This was not the original intention of EPA's comment.

Page 17, Section 4.1.2, Fish Dietary TRVs and Tables 4-3 and 4-4: No extrapolation factors were applied to develop NOECs from LOECs. This is necessary for the use of the dietary approach. Specific protocol should be followed in the development of the dietary TRVs. Previous comments from EPA have emphasized the need to develop dose information for fish and not rely on concentration based TRVs. The concentration only comparison should be omitted and toxicity data from various toxicity endpoints should be converted to an exposure dose in mg/kg/day. This information can be compiled, and fit to a cumulative distribution function similar to what was done for the aquatic TRV development.

Menzie-Cura and the US Army Corps of Engineers evaluated a number of PAH studies to develop dietary TRVs for PAHs. In the Menzie-Cura example, a database of NOAELs and LOAELs was compiled consisting of PAH toxicity data. These included PAH toxicity data from 15 studies on 8 species, and included various life stages (larvae, fry, juvenile, adult), various compounds (e.g., benzo(a)pyrene, DMBA, fluoranthene, anthracene, and phenanthrene), various routes of exposure (water, diet, injection), various exposure durations (single injections, weeks, months), and various toxicity endpoints (hepatic lesions, growth, immunological, reproductive). Water concentrations were converted to an exposure metric (mg/kg/d) using the Arnot and Gobas equations (2004). The data was then fit to a cumulative distribution function, and the geometric mean of the NOAEL and LOAEL was calculated for each study to estimate NOAELs where they were not reported. The data were fit to a log-logistic cumulative distribution function, which was then used to estimate protective doses below which adverse effects in most fish are unlikely. This analysis developed benchmark doses of about 0.01 to 0.2 mg/kg/day to represent doses below which toxic effects are not expected for most fish. This range is below the selected PAH LOAEL TRV reported in Table 4-3 of 1.9 (mg/kg/d). The goal should be to develop dietary NOAELs for fish that are protective of all endpoints. The methodology outlined

above seems like a good model to work from. This model can be carried forward to the other chemicals for which the dietary approach was used (e.g. Table 4-3 and 4-4).

For tables 4-3 and 4-4 the final TRV selection was not clear. The selected concentration based TRVs differ from the dose TRVs. The concentration values should be converted to a dose and used in the TRV development.

Page 18, Section 4.2, Summary of TRV Analysis for Wildlife: Tissue residue concentrations in egg are presented here as a secondary line of evidence for birds. Tissue residue concentrations in bird eggs is considered one of the primary lines of evidence for certain contaminants, such as dioxin and dioxin-like chemicals, PCBs and DDE.

Pages 18 and 19, Section 4.2.3, Bird Egg TRVs: The PRE states that using TRVs based on bird egg concentrations will be used as a secondary line of evidence. For dioxins, dioxin-like chemicals, PCBs, and DDE, the bird egg TRVs should be the primarily line of evidence based on the mode of action of these chemicals (the mode of action is on the developing embryo, not the adult, so a dietary approach is not appropriate and should not even be conducted in the risk assessment for these chemicals). The LWG should not rely on overly conservative egg TRVs (such as those derived for dioxin-like chemicals) or biomagnification factors (BMFs), and more realistic values for these parameters should be used in the next risk evaluation phase. The BMFs for both eagles and ospreys used in the analysis should be based primarily on those derived for ospreys in the Willamette River. BMFs from the Columbia River eagle studies are likely overly conservative if used to represent the lower Willamette River due to the sediment and food chain differences between the two Rivers.

Page 20, Section 5.0, Exposure Characterization: The text here should say that exposure was not estimated for all the chemicals detected in Round 2 sediment, as listed in table E-1

Page 21, Section 5.1.1.1, Site-specific relationship between sediment and tissue chemical concentrations: Due to the lack of information presented in the PRE, EPA is unable to replicate the analysis between sediment and tissue chemical concentrations. However, any additional evaluation of the relationship between sediment and tissue chemical concentrations should incorporate the results of the sediment and benthic tissue sampling that took place in the fall of 2005.

Page 24, Section 5.1.3.2, Selected BMFs: Maximum BMFs derived from all studies should not be used. Rather, the most representative BMFs for the region and species which, in this case, will be from data on ospreys in the Willamette River, should be utilized.

Page 24, Section 5.1.2, BCF Retrieval: *Why were BCFs only retrieved for Aroclors 1260, 1254, 1242, and 1016? Water BCFs should be based on the individual congeners, and not on sums.*

Page 25, Section 5.2.1.1, Tissue Approach: The text states "in the risk characterization 95% UCLs of chemical concentrations in tissue were used to represent less conservative EPCs". Evaluations of tissue residues for the purposes of evaluating fish health should not include a mean value calculated from composite means, especially of the composites were taken over an

area larger than a localized population. Although this may provide information about the range of risk at the site, future evaluations should compare each composite individually against appropriate TRV, and the areas exceeding reported. This represents a key spatial scale issue that was outlined in the March 15, 2006 Ecological Risk Assessment Decision Framework and must be resolved prior to conducing the baseline ERA. Development of 95% UCLs on the mean of composite samples may be more appropriate when evaluating the risk to those species feeding on the fish over large areas (e.g. human health, eagle, osprey, mink).

Page 26, Equation 5-2, Estimating Benthic Invertebrate Tissue Conc.: It is unclear why the mean OC-normalized surface sediment concentration was used to represent surface sediment concentration. The range should be represented – e.g. there may be significant area above the mean value used here, and this would underestimate benthic tissue concentration in some areas.

Question: Tissue concentrations are lipid normalized. Do the BSAFs presented in the l

Page 26, Equation 5-3, Estimating Invertebrate Tissue Concentrations: Because the PRE is a fundamentally a screening step, it is inappropriate to use the 95% UCL of the mean to estimate water column invertebrate concentrations. The maximum surface water concentration should be used as a screening step.

Page 28, Equation 5-4, Estimating Dietary EPC based on Diet Concentration: It is difficult to difficult to represent a dietary EPC based on a contaminant concentration without expressing it as a dose, as the amount of sediment ingested as a proportion of the diet invariably effects the concentrations received in the food and this doesn't make sense. EPA recommends expressing all TRVs as dose and only use equation 5-5 to represent the dietary exposure for fish.

Page 30, Section 5.2.1.3, Dietary Assumptions, Fish: The average body weight (BW) is used to estimate food ingestion rates (FIRs) for each fish. The range of body weights should be reported; implications for the potential range of fish body weights on risk estimates should be discussed.

How will this be addressed in BERA? It seems we should be looking at a mean body weight or body weight distribution.

Pages 30-36, Section 5.2.1.3, Dietary Tissue Assumptions: Taking the maximum of potential prey items is conservative and is an appropriate screening step. However, further refinement of the dietary approach is required prior to initiation of the baseline ERA. For example, it is unlikely that smallmouth bass are not feeding on brown bullhead, northern pikeminnow, carp or black crappie at the size classes samples as described in the dietary assumptions for smallmouth bass (page 35). In this instance, it is more relevant to have sculpin and crayfish represent smallmouth bass diet. Alternative dietary scenarios could be evaluated – for example one scenario could assume smallmouth bass feed primarily on crayfish while another could assume smallmouth bass feed primarily on sculpin. Risk estimates could then be compared to ensure that the risk estimates and subsequent target cleanup levels are appropriately conservative.

Page 36, Section 5.2.2, Benthic Invertebrate Exposure Assessment: There is a statement here that the bioassay testing (direct toxicity testing) will be the primary line of evidence for evaluating risks to the benthic community, that the other "qualitative" lines of evidence may include "assessment of risks via the transition zone and surface water exposure pathways." Exposure to transition zone water and surface water represent different exposure pathways and should be evaluated as such. EPA agrees that the risk to the benthic community may also be evaluated through application of a benthic tissue TRV.

Page 37, Section 5.2.2, Benthic Exposure Assessment: 95% UCLs (on the mean) are appropriate when a mobile receptor is exposed to a media or prey. As it is feeding it will be exposed to an average concentration (represented by a 95% UCL). However, to assess risk to a species of concern, tissue residue values should not be compared to a mean of tissue residue concentrations, because the risk assessment must consider the spatial distribution of contamination and concomitant risk. In the baseline ERA, each sample composite should be compared individually to a TRV, and not averaged over the site.

Page 37, Section 5.3, Exposure Characterization: Wildlife: For most receptors, average exposure values were used to calculate risk estimates (e.g. body weight, ingestion rates). Male and female differences could be important in determining sensitivity. A range of risk estimates based on male and female body weights and ingestion rates could be presented in the baseline report. It might be more appropriate to just use mass and other exposure parameters for birds and mammals based only on the female, as females typically represent the most sensitive parameters to contaminants.

Page 38, Section 5.3.1.2, Dietary Exposure Doses: EPA was unable to replicate the dietary doses presented. It is unclear what sediment concentrations were utilized in the equation.

Pages 39-47, Section 5.3.2, Dietary Assumptions: Average percent moisture values for Round 1 tissue samples were used for all receptors to estimate wet weight food concentration in order to calculate dose. This included all tissue from round 1 including invertebrates and fish. It is inappropriate to use the average moisture content to represent fish and invertebrates in the dietary approach. Percent moisture data should be evaluated and reported on a species-specific level in the risk assessment. Percent moisture should be calculated for different species, and carried through the dietary dose equations. This is a sensitive parameter in this conversion, since it will vary the exposure concentration.

Page 46, Section 5.3.2.6, River Otter: The carnivorous mammal prey that should be used here would be a Portland Harbor specific fish % moisture value from round 1.

Page 49, Section 6.0, Risk Characterization: The text states "the NOAEL-based HQs less than a value of 1 are assumed to represent acceptable levels of exposure and risk for the receptor / pathway / COPC combinations represented by the analysis." EPA notes that this statement is based on Round 1 tissue data, Round 1 and Round 2 sediment data and one round of surface water data. Additional data collection efforts (e.g., Round 2 benthic tissue data, Round sediment and tissue data and subsequent rounds of surface water data) may identify additional chemicals that contribute to site risk.

Page 49, Section 6.0, Risk Characterization. As suggested earlier, do not carry forward in subsequent risk evaluations the information and analysis related to the dietary approach as represented by a concentration; rather, use only the dietary approach represented by a dose.

Page 50, Section 6.1.1, Largescale Sucker: Sediment from the harbor contains PAHs within a range that has been associated in other studies with lesions in fish. In subsequent rounds of sampling, collection of data on external abnormalities on fish is warranted as an additional line of evidence for evaluating PAH exposure and effects. Some lesions and abnormalities are detrimental to fish, and data would need to be made available indicating specific lesions or abnormalities are not detrimental on the individual or population level in order to not be included in the risk assessment. The individual level is important when considering PAH effects to salmonids and lamprey.

Page 51, Section 6.1.2, White Sturgeon: A dietary approach as represented by a should be included as option for assessing risk to white sturgeon.

Page 55, Section 6.1.12, Initial List of Aquatic Organism Chemicals of Potential Concern: The PRE states that "Five additional chemicals were identified with HQs greater than 1 based on white sturgeon and adult lamprey tissue: antimony, arsenic, cadmium, silver and selenium. Although additional data collection efforts focused on sturgeon and lamprey are expected to completed as part of Round 3, these chemicals should not be eliminated as COPCs at this time.

Page 60, Section 6.3.1.1, Use of 95% UCLs of Sediment Concentrations to Calculate Tissue Residue EPC: 95% UCLs for fish tissue are appropriate for developing exposure point concentrations (prey) for higher trophic level organisms (e.g. human health, osprey, eagle, mink) feeding on fish. However, calculating 95% UCL on a mean of composite (average) data may not be protective of fish populations from localized areas. Each individual composite sample (which actually represents a mean or average) should be compared to a TRV directly. Further discussion regarding the scale of exposure and development of exposure point concentrations for receptors that are exposed on a smaller scale than the entire site is required prior to initiation of the baseline ERA.

Page 61, Section 6.3.2.2, Use of 95% UCLs of sediment concentrations to calculate dietary EPCs: It is unlikely that some fish species (e.g. sculpin, bass) would be exposed to a mean of the entire 9 miles of ISA. Therefore, meaningful exposure areas should be used if determining 95% UCLs of a sediment area. For smallmouth bass, this area may be as little as ¼ of a mile. Also, sediment exposure areas may not include channel areas, which may not be likely habitat for some species. As stated above, further discussion regarding the scale of exposure and development of exposure point concentrations for receptors that are exposed on a smaller scale than the entire site is required prior to initiation of the baseline ERA.

Page 63, Section 6.3.3.2, Use of 95% UCLs of Sediment Concentrations to Calculate Dietary EPCs: Representative soil within the range of receptor and not soil from the entire site should be used to represent an EPC in the baseline ERA. It is unlikely that some receptors (e.g. sandpiper) would be exposed to a mean concentration over the entire 9 miles of Portland Harbor Site. Using the entire range may "dilute" what the receptor is actually exposed to.

Page 64, Section 7.0, Identification of Uncertainties: Although overly conservative biomagnification factors may have been used to estimate egg concentrations, further refinement of this approach in the baseline ERA should lead to more realistic results.

Page 65, Section 8.0, Conclusions: How were the many chemicals that didn't have a calculated NOAEL (some had LOAELs), fit into the statement that "COIs for which screening-level exposure estimates did not exceed NOAEL-based TRVs are not likely to represent unacceptable risk for the pathways evaluated"... handled? Were they all "screened in"?

Not sure I agree with above comment. Has anyone looked at TRVs presented in appendix B (TRV TM) and compared the selected NOEC/LOECs with the TRVs used in the screening step as directed by EPA (Table 4-1)?

Section 9, Data Gaps and Recommendations for Additional Information: For most receptors, 100% site use will be a reasonable estimate of their range. For higher trophic level feeders, "use" areas should be determined based on the breeding range (especially when assessing contaminants that impact reproductive success or early life stages). Any data available indicating a receptor uses less than 100% of the site will need to be reviewed and approved by the EPA team before incorporating into the risk assessment. In addition, the exposure areas determined in the PRE are broad and need to be refined for many receptors based on range and how a receptor uses the habitat. Maximum concentrations or 95% UCL may not be appropriate to represent many receptors, so further discussion will be needed regarding how to best spatially average the exposure areas.

Table 2-1, PH Work Plan Assessment Endpoint Table: EPA made modifications to the assessment endpoint table as presented in our December 2, 2005 Identification of Round 3 Data Gaps Memo. Finalization of the assessment endpoint table in the context of the LWG's March 15, 2006 Ecological Risk Assessment Decision Framework is required.

Under largescale sucker, the text that LWG inserted states "In addition, field notes of observed external abnormalities in fish collected from the site have been provided to EPA." It should be noted that contrary to EPA's direction, data on fish lesions and abnormalities in earlier rounds of sampling were not collected consistently by the field crew and some field notes were unintelligible, and therefore do not provide reliable information for decision making. Additional collection of external abnormalities on fish is warranted in subsequent rounds to evaluate this line of evidence for PAH exposure and effects.

Table 4-4- Dietary Fish TRVs: NOECs are not reported for tributyltin, total PAHs, total PCBs and total DDT. NOECs should be estimated from LOECs through application of an appropriate conversion factor for TRV development.

Table 4-6, Wildlife Dietary TRVs and Table 4-7, Mammal Dietary TRVs: *To my knowledge, these still need to be reviewed by EPA. It is unclear if the TRV values were reviewed by Parametrix to ensure they were appropriately protective, and that the literature review included all relevant studies.*

Table 5-7, Estimated Invert Tissue Residues: As stated in our comment regarding Equation 5-3 above, 95% UCL of the mean surface water concentrations should not be utilized. A range of modeled water-column invert tissue concentration should be presented and used in the risk estimates. For the PRE, the maximum water concentration should be be used.

Table 5-9, Fish Dietary EPCs: It is important to agree on an appropriate way to move forward from the maximum sediment concentration to more of an area-weighted average. Consensus on an appropriate area of exposure (e.g. home range) for each receptor before the next submittal is necessary. The 95% UCL on the mean will not be appropriate for all receptors, as it would not be likely that they would be exposed to the 9 plus miles of ISA.

Table 5-14, Bird and mammal dietary prey assumptions: Prey % moisture characteristics should be specific to the prey of each receptor. For example, for osprey the % moisture should be the average of the species listed, and shouldn't include clams.

Table 5-15, Bird Dietary Exposure Doses: Several food ingestion rates are calculated using the literature to define the % moisture of the diets, and others used the Round 1 tissue average concentration. For determining a site exposure dose we are converting a dry weight (dw) food ingestion rate and converting it to a wet weight and should assume they are feeding from the Willamette, and use appropriate % moisture data from prey items identified in the Round 1 tissue collection. For the development of TRVs we should be using the % moisture from the study the TRV was developed.

Appendix B, TRVs: *Why were NOEC values selected that are higher than a relevant LOEC (e.g. see Figure 17 for copper)? Why weren't LOECs selected in some cases (e.g. copper)?*

Appendix C, Statistical Analysis of Site-Specific Relationship Between Tissue and Sediment Chemical Concentrations: This section is a clearly written and understandable synthesis of a somewhat complex issue. However, there is not enough information presented here to replicate the analysis. The BSAF approach described is currently limited by small sample size or limited number of samples where contaminants in both sediment and tissue were detected (e.g., DDE was only detected in both the sediment and sculpin tissue of only six samples). An additional evaluation should be conducted to examine BSAF relationships based on only the samples detected in both tissue and sediment (i.e., discarding samples with non-detected values in both tissue and sediment) to see if a better relationship can be identified. Additional data on sculpin and other tissue during the Round 3 collection will hopefully provide better insight on the relationship between sediment and tissue values, as the BSAFs currently identified for DDE and PCBs appear much lower than literature values. Also, additional analysis of PCB BSAFs for sculpin should be conducted by evaluating only the total PCBs as Aroclor 1260 and by using the total PCB values calculated by summing the congeners. These may represent better relationships than summing Aroclors (which can include "double" counting of overlapping congeners).

Appendix C, Page 2-3: Relationships between sediment and tissue should be tested for significance at an alpha level of 0.1. This was commented on previous versions of the PRE approach. It appears the final PRE did not follow previous comments made, or the PRE

Approach Technical Memorandum presented in Appendix A, indicates that an alpha of 0.1 will be used. Only relationships with a significance of 0.05 were pursued further; this limitation could overlook significant relationships and other contaminants. The nature of the correlation between tissue and sediment concentrations should be investigated using scatter-plots and regression analysis for those that had a significance of 0.1. Also, for the regression relationships, it should not be concluded, "if the slope (m) is not significantly different from zero (with an alpha of 0.5) then the linear relationship between log-tissue and log-sediment is not significant and log-tissue can be most efficiently approximated using the geometric mean of tissue concentrations rather than modeled as a function of sediment concentrations".

Appendix C, Section 3.3, DDE in Sculpin Tissue: For BSAF analysis, non-detect values should not be treated as ½ the detection limit. The data gap here should be filled with more tissue sampling. In this case, 11 sediment and 5 tissue samples were non-detect (we need to know if these were elevated detection limits), and only 6 samples actually had co-located detections of sediment and tissue. The non-detects in this case is likely to skew the development of the relationship.

Appendix E, Evaluation of Uncertainties, Section 3.1.5, Performance of Additional Risk Analysis: The analysis of human health fish for ecological risk showed higher HQs, which may be important for some that showed up based one line of evidence (e.g. zinc, chromium and cadmium). For example, zinc was just over an HQ of one for juvenile Chinook, and close for all other species 0.6 to 0.93. Chromium shows up here, and in the 5th percentile analysis (LOEC), which as one order of magnitude lower than the Appendix B NOEC and LOEC.

Appendix E, Section 3.2, Dietary Approach: Given the HQ values for TBT in this report, TBT should be analyzed for in future fish tissue sampling. This is a data gap in that right now we only have the dietary approach as one line of evidence.

Appendix E, Section 3.2.3, Availability of tox data for dietary TRVs: We should be calculating a NOAC from a LOEC if it is available. Dietary LOECs may have underestimated dietary risk and this should be revised.

Appendix E, Section 3.2.4, Maximum sed / tissue conc: The maximum concentration risk may be indicative of localized risk, which may be relevant for some receptors. This should be discussed as to where and how big these areas are likely to be within the ISA.

Appendix E, Section 4.1, Dietary Approach: The use of 100% site use for receptors in the risk assessment is listed as an uncertainty. For any receptor where 100% site use is not used, the data available to change the site use will need to be reviewed. We should be working on these issues before the next submittal.

Appendix E, Section 4.1.2, Availability of Tox Data for dietary TRVs: Where TRVs are not available, the use of a surrogate should be explored.

Appendix E, Section 4.1.3: The use of 95% UCL sediment concentrations is cited as potentially being more realistic. Were these calculated using relevant organism foraging areas (e.g. near shore versus channel) or were all sediment samples used to develop that mean?

Appendix E, Table E-1: Table E-1 summarizes chemicals that were identified in Round 2 sampling that were not found in Round 1 sediment or tissue sampling. Because TRVs were developed only for COIs found in Round 1 sampling, Appendix E of the PRE acknowledges that "These chemicals will be added as COIs in the next iterations of the ERA." Contaminant data from Round 2 not evaluated in the PRE and future Round 3 data collection efforts should be used to re-evaluate exposure and risk to ecological receptors.

Appendix E, Table E-4: What does the "a" footnote mean? Is it based on detection limit?

Appendix E, Table E-5: How is "risk driver" defined here? How were determinations of "underestimate" and "overestimate" made?

Appendix E: Section 2.2., page 2, under "Fish." It is inappropriate to use other fish as a surrogate for lamprey, but at least the TRVs derived for the most sensitive fish can be used to estimate potential risk to lamprey (along with describing the assumptions and uncertainty around the estimate).

Appendix E, section 4.2.1: Explain why egg injection studies may overestimate risk as opposed to underestimating risk.

Appendix E, section 4.2.3: For the next phase of the risk assessment, do not use the most conservative BMF values. Rather, use values that most represent the site (i.e., BMFs derived for the Willamette River fauna). The bird egg approach should be modified to use more realistic data (such as BMFs and TRV values), which should improve the reliability of the approach.

Supplemental Comments on TRV Development

The comments presented below address TRVs as utilized in the PRE. The comments are designed to assess with the TRVs utilized in the PRE are consistent with EPA direction as outlined in our comments dated June 10, 2005 and whether they are adequately conservative relative to the toxicity data compiled in the TRV TM (Appendix B of the PRE). Additional discussions are needed with LWG and EPA and its partners to evaluate the differences obtained between the TRV derivation methods (e.g., differences among TRV results based on the 5th percentile LOEC and the modeled methods).

Table 1 compares the NOEC and LOEC TRVs from the TRV TM with the TRVs used in the PRE. As shown, the PRE TRV for several chemicals is greater than the TRV TM LOEC. The TRVs for these chemicals are each briefly addressed below.

<u>Aquatic Life Tissue Residue TRVs</u>

- **2,3,7,8-TCDD:** The PRE TRV of 90 pg/g is 46 times greater than the TRV LOEC of 1.95 pg/g (Table 2). This occurs because the lowest LOEC of 1.95 pg/g is much lower than the other 2,3,7,8-TCDD LOECs and the availability of 34 fish whole body LOECs (per Table 1-1, Attachment 1, Appendix B of the PRE) results in a 5th percentile that is greater than the lowest LOEC. Although from a strictly numeric perspective the PRE TRV is less conservative than the LOEC TRV, whether the PRE TRV is sufficiently conservative for the PRE is a risk management decision. Many of the whole body NOEC and LOEC values for 2,3,7,8-TCDD tabulated in the TRV TM, including the lowest LOEC of 1.95 pg/g, were actually estimated from 2,3,7,8-TCDD concentrations measured in fish eggs. However, the lowest *measured* whole body NOEC and LOEC values were 46 and 85 pg/g, respectively, for lake trout. Accordingly, it could be argued that the PRE TRV of 90 pg/g is under-conservative for a screen, although it does appear that the PRE TRV is consistent with the approach outlined by EPA.

- **Total PCBs:** The PRE TRV of 720 µg/kg is slightly greater than the LOEC TRV of 520 µg/kg (Table 2). In Table 1-1 (Attachment 1, Appendix B of PRE), ≥20 PCB LOECs are included, which resulted in the 5th percentile LOEC being greater than the lowest measured LOEC. This approach appears to be consistent with EPA guidance for developing PRE TRVs. However, we do have one question concerning the lowest PCB LOEC. Table 1-1 shows a LOEC of 150 µg/kg, which is less than the LOEC TRV of 520 µg/kg. The text in Appendix B of the PRE states that whole body tissue residue LOECs for PCB Aroclor mixtures ranged from 520-645,000 µg/kg and that "a lower LOEC of 0.15 mg/kg [150 µg/kg] based on [an] injected dose was reported in Folmar et al. (1982) in which yearling coho salmon survival was reduced; however, no tissue residue concentrations were measured in the study." A similar comment is included in the notes for this study in Table 1-1, but perhaps inclusion of this study in the table creates confusion if it does not meet minimum data requirements for TRV development.

- **Copper:** The PRE TRV of 3.1 mg/kg is the 5th percentile LOEC from Dyer et al. (2000), which is almost two times greater than the TRV LOEC of 1.71 mg/kg (Table 2). As discussed above, the PRE did not appear to entirely follow EPA's approach for PRE TRV development, as the Dyer et al. (2000) 5th percentile values are intended to supplement the TRVs developed from the LWGs extensive literature review. Given that the number of LOECs was between 8-19, EPA's approach would also have identified the lowest LOEC of 1.71 mg/kg as a PRE TRV for copper. Technically, EPA's guidance to the LWG would have also required calculating a whole body tissue residue as a function of the chronic copper criterion and a BCF, and then using the lower of this value or the lowest LOEC as the PRE TRV. It is not clear whether the LWG followed this step.

- **Mercury:** The PRE TRV of 0.46 mg/kg is from Dyer et al. (2000) and is two times greater than the LOEC TRV of 0.23 mg/kg (Table 2). Given that 20 LOEC values are reported in Table 1-1 (Attachment, Appendix B of PRE), it appears that a 5th percentile LOEC could have been calculated by the LWG, which likely would have been near 0.23 mg/kg since the lowest LOEC out of 20 values approximates the 5th percentile (depending on the calculation used).

- **Selenium:** The PRE TRV of 1.1 mg/kg is from Dyer et al. (2000) and is almost two times greater than the LOEC TRV of 0.68 mg/kg (Table 2). According to Appendix B of the PRE, 14 LOECs were considered for development of the LOEC TRV. Following EPA's guidance for PRE TRV development, therefore, the LOEC TRV of 0.68 mg/kg should probably also had been considered in the PRE. Although perhaps beyond the scope of this technical memorandum, we suggest that both TRVs (0.68 and 1.1 mg/kg wet wt.) are overly conservative values. We can discuss this with you further if you wish to evaluate this TRV more closely.

  In addition, the TRV LOEC of 0.68 mg/kg reported in Appendix B appears to be erroneous. Cleveland et al. (1993) is cited as the source of this value; however, the value of 0.68 actually refers to the aqueous selenium concentration, in units of mg/L, to which the test fish (bluegill) were exposed. The whole body selenium concentration in the bluegill exposed to an aqueous selenium concentration of 0.68 mg/L was 5 mg/kg dry wt. (or 1 mg/kg wet wt. assuming a moisture content of 80%). Further, it should be noted that use of water-based selenium exposures to derive a fish tissue-based toxicity value is generally not relevant to a field situation where fish are primarily exposed to selenium via the diet. As noted, an aqueous selenium concentration of 0.68 mg/L was required to achieve a tissue selenium concentration that was toxic to bluegill. This is much greater than the water selenium concentrations typically required to achieve a food chain selenium concentration that is toxic to fish. For example, 0.68 mg/L is 136 times greater than the USEPA's current chronic ambient water quality criterion for selenium of 0.005 mg/L. The whole body selenium TRVs for fish should, thus, probably be revisited.

- **Dieldrin, endosulfan (total), and endrin:** The PRE TRVs for all three of these organochlorine pesticides are from Dyer et al. (2000) and all are greater than the LOEC TRVs from the TRV TM. Strictly interpreting EPA's guidance for PRE TRV development, the LOEC TRVs should probably also have been considered in the PRE. Because the dieldrin and endrin criteria are based on FDA action levels, it does not make sense to estimate a tissue residue TRV from their criteria values and BCFs (per our general comment above). However, this approach may be possible for endosulfan.

Fish Dietary TRVs

As noted in the PRE, EPA did not provide any direction on development of dietary TRVs for fish and we could, thus, not evaluate whether the dietary TRVs identified in the PRE are consistent with EPA guidance. However, we note that both NOECs and LOECs were used in the PRE to develop dietary TRVs, but LOECs were preferred in development of tissue residue TRVs. Use of LOECs vs. NOECs is a risk management decision, but perhaps the basis for not also considering NOECs to derive tissue residue TRVs should be discussed.

Avian and Mammalian Dietary TRVs

The EPA's review of the initial TRV Selection Technical Memorandum resulted in three primary comments with respect to wildlife TRVs:

(1) "TRVs for wildlife should be reported as dietary doses and as concentrations in eggs for bird receptors." The LWG agreed to develop bird egg TRVs for dioxins, PCBs, DDE, and possibly mercury, and that for remaining chemicals only dietary dose-based TRVs would be developed for birds.
(2) "The process for selecting TRVs for birds should include field study evaluations and lab studies using egg injection techniques."
(3) "NOAELs can be derived from LOAELs using uncertainty factors and vice versa. Uncertainty factors should be used in order to quantitatively evaluate risk."

We compared the dietary NOAELs and LOAELs for birds and mammals used in the PRE to those in the TRV TM (Appendix B of the PRE). Consistent with EPA's comment, chronic NOAELs were either estimated from effect levels when NOAELs were lacking, or NOAELs were adjusted downward if the exposure was less than chronic (e.g., acute or subchronic). The following summarizes the avian and mammalian PRE TRVs that were adjusted from those in the TRV TM:

- **Birds:** (1) chronic NOAEL estimated from chronic LOAEL using an uncertainty factor of 10 (mercury, PAHs, DDD, DDT, endrin) ; (2) chronic NOAEL estimated from acute LD50 using an uncertainty factor of 100 (thallium, heptachlor); (3) chronic NOAEL estimated from subchronic NOAEL using an uncertainty factor of 10 (zinc, hexachlorobenzene, pentachlorophenol); and (4) chronic NOAEL estimated from acute NOAEL using an uncertainty factor of 30 (methoxychlor, acetone).

- **Mammals:** (1) chronic NOAEL estimated from chronic LOAEL using an uncertainty factor of 10 (2,3,7,8-TCDD, PCBs, dieldrin, endrin, hexachlorobenzene, methoxychlor, Mirex, dibutyl phthalate) and (2) chronic NOAEL estimated from subchronic NOAEL using an uncertainty factor of 10 (antimony, selenium, thallium, naphthalene, 2-methylnaphthalene, beta-HCH, trans-nonachlor, 2,4-dimethylphenol, 1,4-dichlorobenzene, acetone).

Thus, more conservative avian and mammalian TRVs were developed for several chemicals in the PRE. We also compared the dietary wildlife TRVs used in the PRE to the toxicity data provided in Tables 1-4 and 1-5 in Attachment B-1 of Appendix B to the PRE. Overall, it appears that TRVs selected were the most conservative of those from acceptable studies and that the PRE TRVs are reflective of EPA's comments for deriving adequately conservative dietary TRVs.

Avian Egg TRVs

The description of egg TRVs and dioxin-like compounds is a very good and forthright description of the available literature. As noted in this section, the selected TRVs for dioxin-like compounds in bird eggs are very conservative. TRVs derived for non-domestic species more specific to the Willamette River should be used for the next phase of the risk assessment, or a species sensitivity distribution should be derived incorporating laboratory testing on chickens and other species and field testing on wildlife. This process should be discussed and agreed to with EPA and its partners prior to the next phase of the risk assessment. This also applies to the PCB and DDE sections.

Avian egg TRVs were developed for 2,3,7,8-TCDD, PCBs, DDE, and mercury. Per EPA's comments, the LWG considered field data in addition to laboratory data for deriving bird egg TRVs. Given the egg-based toxicity data provided in Tables 1-5 and 1-6 of Attachment B-1, Appendix B of the PRE, the egg TRVs used in the PRE appear to be conservative values. The 2,3,7,8-TCDD LOAEL of 0.01 ng/g is likely conservative for the PRE, as this egg concentration was associated with abnormalities in 1 of 6 chicks, while no abnormalities were observed in control chicks. Consequently, the sample sizes were small, making identification of effect levels difficult. For comparison, Table 1-5 included another egg LOAEL of 0.122 ng/g for chickens exposed to 2,3,7,8-TCDD in a more comprehensive study, which is an order of magnitude greater than the PRE TRV.

Table 1. Comparison of whole body fish tissue-based TRVs from the TRV TM versus those used in the PRE.

		TRV TM		PRE	
Analyte	**Units[1]**	**NOEC**	**LOEC**	**TRV**	**Source**
2,3,7,8-TCDD	pg/g	-	1.95	90	LWG TRV TM (App. B; 5th percentile literature LOECs)
PCBs, total	µg/kg	-	520	720	LWG TRV TM (App. B; 5th percentile literature LOECs)
Antimony	mg/kg			0.03	Estimated from AWQC and BCF
Arsenic	mg/kg	1.0 [a]	1.7 [a]	1.7	Dyer et al. (2000) (5th percentile literature LOECs)
Cadmium	mg/kg	0.038 [a]	0.090 [a]	0.09	LWG TRV TM (App. B; 5th percentile literature LOECs)
Chromium (Cr VI)	mg/kg	5.5	8.7	0.69	Dyer et al. (2000) (5th percentile literature LOECs)
Copper	mg/kg	1.17	1.71	3.1	Dyer et al. (2000) (5th percentile literature LOECs)
Lead	mg/kg	2.54	4.02	2.2	Dyer et al. (2000) (5th percentile literature LOECs)
Mercury	mg/kg	-	0.23	0.46	Dyer et al. (2000) (5th percentile literature LOECs)
Nickel	mg/kg	-	-	18.4	Dyer et al. (2000) (5th percentile literature LOECs)
Selenium	mg/kg	0.6	0.68	1.1	Dyer et al. (2000) (5th percentile literature LOECs)
Silver	mg/kg	0.06	-	0.27	Dyer et al. (2000) (5th percentile literature LOECs)
Thallium	mg/kg	2.72	-	4.6	Estimated from AWQC and BCF
Zinc	mg/kg	34	40	27	Dyer et al. (2000) (5th percentile literature LOECs)
Tributyltin	µg/kg	-	-	49.9	Estimated from AWQC and BCF
DDD, 4,4'-	µg/kg	-	-	54	Estimated from AWQC and BCF
DDE, 4,4'-	µg/kg	-	-	1,000	Dyer et al. (2000) (5th percentile literature LOECs)
DDT, 4,4'-	µg/kg	-	-	470	Dyer et al. (2000) (5th percentile literature LOECs)
DDT, total	µg/kg	1,800	1,800	290	LWG TRV TM (App. B; 5th percentile literature LOECs)
Hexachlorocyclohexane, alpha	µg/kg	-	-	4.9	Estimated from AWQC and BCF
Hexachlorocyclohexane, beta	µg/kg	-	-	4.9	Estimated from AWQC and BCF
Hexachlorocyclohexane, gamma (Lindane)	µg/kg	-	79,000	23	Dyer et al. (2000) (5th percentile literature LOECs)
Hexachlorocyclohexane, delta	µg/kg	-	-	4.9	Estimated from AWQC and BCF
Chlordane, total	µg/kg	710 [a]	1,400 [a]	550	Dyer et al. (2000) (5th percentile literature LOECs)
Dieldrin	µg/kg	120	200	220	Dyer et al. (2000) (5th percentile literature LOECs)
Endosulfan, alpha	µg/kg	-	-	73	Dyer et al. (2000) (5th percentile literature LOECs)
Endosulfan, beta	µg/kg	-	-	73	Dyer et al. (2000) (5th percentile literature LOECs)

		TRV TM		PRE	
Analyte	**Units[1]**	**NOEC**	**LOEC**	**TRV**	**Source**
Endosulfan, total	µg/kg	-	31	73	Dyer et al. (2000) (5th percentile literature LOECs)
Endosulfan sulfate	µg/kg	-	-	73	Dyer et al. (2000) (5th percentile literature LOECs)
Endrin	µg/kg	-	11.5	25	Dyer et al. (2000) (5th percentile literature LOECs)
Endrin aldehyde	µg/kg	-	-	25	Dyer et al. (2000) (5th percentile literature LOECs)
Heptachlor	µg/kg	-	1,500	60	Estimated from AWQC and BCF
Heptachlor epoxide	µg/kg	-	800	55	Estimated from AWQC and BCF
Hexachlorobenzene	µg/kg	468	-	490	Dyer et al. (2000) (5th percentile literature LOECs)
Methoxychlor	µg/kg	50	300	200	Dyer et al. (2000) (5th percentile literature LOECs)
Hexachlorobutadiene	µg/kg	-	20,000	26	Estimated from AWQC and BCF
Hexachloroethane	µg/kg	-	-	47,000	Estimated from AWQC and BCF
bis(2-ethylhexyl)phthalate	µg/kg	390	10,600	390	Estimated from AWQC and BCF
Di-n-octylphthalate	µg/kg	-	-	41,000	Estimated from AWQC and BCF
4-Methylphenol	µg/kg	-	76,500	76,500	LWG TRV TM (App. B; 5th percentile literature LOECs)

[1] All concentrations are expressed on a wet weight basis.
[a] NOEC and LOEC not from same study.

These comments were offered up by Parametrix to make sure we know what we are asking for:

First are a few general comments before supplying specific comments on whether the TRVs used in the PRE are consistent with EPA guidance.

(1) Application of the hierarchy for identifying PRE TRVs, will almost always result in the 5th percentile LOEC (when ≥20 LOECs are available) greater than the lowest LOEC (depending on the method used to calculate the 5th percentile). Thus, this hierarchical approach does not usually derive the most conservative TRVs. Perhaps EPA is already aware of this and comfortable with the approach, but we want to raise this issue because EPA was specifically concerned that the TRV LOECs from the TRV TM were not adequately conservative for the PRE.

(2) *Multiplication of an AWQC by a BCF to estimate a whole body-based TRV should be done with some caution to ensure that the resulting TRV is reasonable and makes sense. For example, PRE TRVs for 4,4'DDD, heptachlor, and heptachlor epoxide were calculated using this approach, despite that the AWQC used had already been back-calculated from a human health- or wildlife-based toxicity value (Table 1). Thus, application of an aquatic organism-based BCF to a human health- or wildlife-based criterion to calculate a tissue-based TRV protective of aquatic life results in a value that is difficult to define. In such cases an alternative option is use of a final chronic value (FCV) or other aquatic life-based toxicity value.*

(3) EPA's comments to the LWG (June 10, 2005) state that species sensitivity distributions (SSDs) should be developed from the lowest adverse effect residue from each available study and that the use of SSDs will allow EPA to define the proportion of species they want to protect. However, we note that under the EPA's guidance to the LWG multiple LOECs for the same species may be used to develop the SSD, so the SSD will not necessarily provide information on the level of species protection. Ideally, in developing an SSD, a single LOEC would be identified for each species, which could be either a minimum or average LOEC if multiple values are available for a species.

Table 1. Basis of chronic ambient water quality criteria for 4,4'DDD, alpha-, beta-, and gamma-hexachlorocyclohexane, heptachlor, and heptachlor epoxide.

Analyte	Chronic Criterion (µg/L)	Basis
DDD, 4,4'	0.001	Back-calculated from: the lowest maximum permissible tissue concentration of 0.15 mg/kg based on reduced productivity of the brown pelican divided by a geometric mean lipid-normalized BCF of 17,870 and assuming a lipid content of 8%
Heptachlor Heptachlor epoxide	0.0038 [a]	Back-calculated from: the FDA action level of 0.3 mg/kg for edible fish and shellfish by a geometric mean lipid-normalized BCF of 5,222 and assuming a lipid content of 15%

[a] Minor point, but this was identified in the PRE as the marine chronic criterion. This value is actually the freshwater criterion and the marine criterion is 0.0036 µg/L.

FDA = Food and Drug Administration